EXHIBIT 14

Rebuttal Report of Suresh H. Moolgavkar, M.D., Ph.D.

April 6, 2009

In this report, I have been asked to review and comment on the December 2008 expert report of Dr. Whitehouse.

One of Dr. Whitehouse's central theses appears to be that Libby fiber is uniquely toxic and that it has caused, and will continue to cause, serious disease in the general population in the Libby area. The quantitative assessment of the toxicity of any agent requires a proper epidemiological study with good quantitative information on exposure. The cohort of workers at the Libby mines has well-characterized exposures and has been used to assess quantitatively the risks posed by Libby fibers in a number of investigations, most recently by Sullivan (2007). In addition, disease in the general population in the Libby area has been investigated by the Agency for Toxic Substances and Disease Registry (ATSDR). Instead of relying on these studies, Dr. Whitehouse relies on observations on patients in his clinical practice and on deeply flawed studies that he has conducted on his patient population.

Dr. Whitehouse's conclusions are based on improper and unacceptable analyses of various poorly characterized data sets and on anectodal evidence. He relies in particular on a dataset that he established at the Center for Asbestos Related Disease (CARD) at Libby, Montana. This database apparently consists of information on over 1,600 patients with what Dr. Whitehouse calls "asbestos-related disease". Dr. Whitehouse offers no description of how the database was assembled and no evidence that the patient population at CARD is representative of the general population of the Libby area. Thus, any conclusions that might be drawn from this dataset cannot be extrapolated to the Libby area population at large.

CARD mortality study is fatally flawed and provides no reliable information on the toxicity of Libby asbestos

Dr. Whitehouse provides no protocol for his CARD mortality study. In particular, there is no information on how the study was designed, and how the cohort of study subjects was assembled, except that it is some subset of Dr. Whitehouse's patient population. Dr. Whitehouse's comparison of the mortality experience of this cohort of patients with that of the insulators' cohort does not follow established scientific practice and is, therefore, un-interpretable. First, it is totally inappropriate to compare the mortality experience of a cohort of patients who already have asbestos disease with an occupational cohort of asbestos workers. Dr. Whitehouse argues that workers in the insulators' cohort who died must have had asbestos disease. He has no direct evidence to support this claim. And, even if true, he is still comparing mortality in the entire insulators' cohort with mortality in a group of patients with asbestos disease. The appropriate comparison group for the insulators' cohort is clearly the occupational cohort of workers at the Libby mines. This occupational cohort has been investigated in great detail in a number of published studies. I discuss the findings of these studies below.

Second, even if for some reason one wanted to compare the mortality experience of the CARD cohort with that of the insulators' cohort, there are well-established scientific principles that must be used in making such comparisons, which Dr. Whitehouse

completely ignores. For example, age is an important and strong determinant of mortality risk and must be accounted for in any comparison of the mortality experience of two populations. There are standard epidemiological methods for doing this, which Dr. Whitehouse, if he is aware of them, has chosen not to use. Some of these methods, such as age-standardization of rates, are quite straightforward to understand and implement with a hand-held calculator. Others, such as Poisson and Cox regression, require the use of specialized software, but are now commonplace in epidemiological analyses.

Finally, Dr. Whitehouse has absolutely no information on the level of exposure to Libby fiber in his patient population.

Dr. Whitehouse uses the comparison of the mortality experience of his cohort with that of the insulators' cohort to argue that Libby asbestos is highly toxic because similar mortality patterns were seen in the two cohorts with allegedly much lower exposure levels[1] in his patient population. Thus, Dr. Whitehouse argues that Libby fiber is far more toxic than the fibers that the insulators were exposed to. As discussed above, this conclusion simply cannot be drawn from the flawed analyses presented by Dr. Whitehouse. However, the issue of potency of Libby fiber can be addressed with the available data on the occupational cohort at Libby.

As I noted above, the mortality experience of the insulators' cohort should be compared with the mortality experience of the Libby occupational cohort. More generally, it is of interest to view the mortality risks in the Libby cohort within the framework of the mortality risks observed in other occupational cohorts exposed to asbestos. I present this comparison below for three disease end-points, lung cancer, mesothelioma, and non-malignant respiratory disease (NMRD), which includes asbestosis. These comparisons show that the toxicity of Libby fibers lies in the middle of the range of toxicities for asbestos fibers.

Instead of using the well accepted quantitative approaches described below, Dr. Whitehouse bases his opinions on anecdotal evidence. For example, on page 28 of his report, he gives three examples of individuals with minimal exposures to Libby asbestos who he alleges have developed lung cancer, non-malignant respiratory disease (NMRD) and mesothelioma, respectively, as a result of this minimal exposure. The logic of his allegation defies comprehension. As laid out in detail in my previous reports (June 11, 2007; July 31, 2007), a proper quantitative analysis of other exposures including, if possible, the estimation of etiologic fraction, is imperative before any conclusions can be drawn regarding the contribution of Libby fibers to disease causation.

As I have said above, it is possible to develop good quantitative information on the toxicity of Libby asbestos because of the availability of properly conducted epidemiological studies on the occupational cohort at Libby. In addition, the issue of whether environmental or non-occupational exposure to Libby asbestos poses a risk to the general population at Libby has been addressed in studies conducted by the ATSDR. I discuss these studies here with emphasis on the latest findings.

[1] Dr. Whitehouse has no quantitative information on the level of exposure in his patient population.

Lung Cancer

Lung cancer risk in the Libby occupational cohorts can be compared quantitatively with risks in other occupational cohorts using two distinct approaches. The first is based on Standardized Mortality Ratios (SMR) and relative risks (RR) and has been the method used by the EPA. The second approach was developed in Hodgson and Darnton (2000).

The latest comprehensive analyses of all asbestos cohorts with reasonable quantitative information on exposure are presented by Berman and Crump (2008). The results for lung cancer are in table 3. The Libby potency factor[2] for lung cancer, $K_L = 0.0026$, is in the middle of the range of potency factors in the table and approximately equal to the potency factor in the insulators' cohort (0.0028). Thus, for lung cancer mortality, Libby fiber is certainly no more toxic than the fibers the insulators were exposed to. Moreover, the Libby fibers are considerably less toxic than the fibers that the South Carolina Textile Cohort was exposed to, which were predominantly chrysotile fibers. The potency factor of 0.0026 for Libby fiber is about a fourth of the potency factor used by EPA in its 1986 risk assessment for asbestos[3] and implies that the relative risk (RR) of lung cancer associated with a cumulative exposure of 100 f/ml-y of Libby fiber is 1.26, about the same as the RR associated with exposure to second-hand smoke. To put this in context, the RR of lung cancer associated with a 20 pack-year smoking habit is more than 10.

The analyses described above were based on SMRs. One problem with SMR analyses is selection of the appropriate external control group. The U.S. population is generally used as the control. The SMR thus depends upon the choice of control and on the assumption that the rates in the control group are a good representation of the underlying rates in the study population, i.e., the rates that would be expected in the absence of exposure to asbestos. The SMR depends also on the number of exposure categories selected for analyses. In order to get around these problems, methods of analyses based on internal controls can be used. A commonly used approach is Cox proportional hazards regression modeling. Amandus and Wheeler (1987) used this approach in their Libby occupational cohort and estimated a potency factor $K_L = 0.0011$, less than half the potency factor estimated in Berman and Crump. It is interesting that a similar, but somewhat smaller, potency factor (0.0008) was reported in an unpublished ATSDR document (Larson and Phalen, unpublished manuscript) based on an updated Libby cohort, a sub-cohort of which was analyzed by Sullivan (2007). We have recently completed analyses of the Sullivan database. Using the Cox model, we estimate that the potency factor of Libby fiber is 0.0011, identical to the potency reported in Amandus and Wheeler (1987). Loomis et al. (2009) report the same potency factor in the North Carolina textile workers cohort, which was exposed predominantly to chrysotile. These results imply that the RR

[2] These potency factors are estimated on the ASSUMPTION that the standardized mortality ratio (SMR) is a linear function of cumulative exposure without any threshold. These results do not imply that there is non-zero risk at the lowest levels of exposure.

[3] Although EPA has proposed revisions to its current approach to asbestos risk assessment to take account of fiber type and size, the Science Advisory Board (SAB) was critical of the implementation plan the EPA put forward. Therefore, the EPA approach has not been revised.

associated with a cumulative exposure of 100 f/ml-y to Libby fiber is 1.11, far less than that reported with exposure to second-hand smoke. These results also indicate that Libby fiber is no more potent than chrysotile as a lung carcinogen.

A second approach to comparing the potency of Libby fiber with fibers that other occupational cohorts were exposed to is to use the Hodgson-Darnton (H-D) index. With an estimated SMR of 1.7 (Sullivan, 2007) and average cumulative exposure of 91.4 f/ml-y, the H-D index for Libby fiber is 0.77, considerably smaller than the H-D index for crocidolite (Hodgson and Darnton, 2000; table 2) and in the middle of the range for the chrysotile cohorts analyzed in Hodgson and Darnton (2000).

Mesothelioma

As in the case of lung cancer, there are two approaches to comparing potencies of the fibers that various occupational cohorts are exposed to. The first approach adopted by the EPA is based on a model for mesothelioma mortality hazard developed by Peto (1979). Berman and Crump present the potencies (K_M) based on this approach for various cohorts. While they do not compute the potency for Libby fibers, we are able to do so based on the Sullivan dataset. Sullivan reports that there are 15 mesotheliomas in the updated Libby cohort. Using this number and the detailed exposure history of each member of the cohort, we are able to estimate that for the Libby cohort, $K_M = 0.5 \times 10^{-8}$, half the potency factor assumed by EPA in its 1986 risk assessment for asbestos[4]. From table 4 of Berman and Crump (2008), we can see that this estimate of Libby potency lies in the middle of the range of potencies for asbestos fibers, and is much smaller than the potency for crocidolite and somewhat smaller than the potency for amosite.

The second approach is to use the H-D index for mesothelioma. With 15 mesothelioma deaths, 767 total deaths, and average cumulative exposure of 91.4 f/ml-y, the H-D mesothelioma index for Libby is 0.02, again right in the middle of the range reported in Hodgson and Darnton (2000, table 1), considerably smaller than the H-D index for crocidolite, and somewhat smaller than the H-D index for amosite.

Dr. Whitehouse contends, without offering the requisite evidence on other exposures, that environmental exposure to Libby fibers has led to numerous mesothelioma deaths. I find it hard to decipher from his report how many such deaths he is alleging. Exhibit 3 of his report is a paper he wrote on environmental exposures and mesothelioma at Libby (Whitehouse, 2008). In the abstract to that paper he states, "[t]hirty-one cases of mesothelioma resulting from exposure to Libby Asbestos have been identified from Libby, Montana. Eleven cases not previously reported are the subject of this report." This statement would seem to imply that exposure to Libby asbestos led to 42 cases of mesothelioma. In the introduction to the paper Dr. Whitehouse says, "Sullivan [2007] reported that 15 cases of mesothelioma were documented in WRG workers as of 2001. We report an additional 11 cases of mesothelioma that have been documented among non-occupationally exposed people by the Center for Asbestos Related Disease (CARD)

[4] See footnote 3.

in Libby." This statement would seem to imply that exposure to Libby asbestos led to 26 cases of mesothelioma. Finally, in his exhibit 9, he lists 34 cases of mesothelioma.

As mentioned above, and described in detail in previous reports, all exposures must be carefully considered before any conclusions regarding the contribution of Libby fibers to any case of mesothelioma can be drawn. In particular, although rare, mesothelioma can occur without exposure to asbestos. Although Dr. Whitehouse asserts that the age-adjusted rate of such spontaneously occurring mesothelioma is less than 1 per million individuals per year, recent analyses (Moolgavkar et al., 2009) suggest that the age-adjusted rate for pleural mesothelioma is 2-3 per million individuals per year, and that for peritoneal mesothelioma is about 1 per million individuals per year.

Non-malignant respiratory disease (NMRD)

Unlike lung cancer and mesothelioma, it is not possible to compare the potency of Libby fiber for NMRD with other fibers because this information for the other fibers is not available. The focus of most quantitative analyses of occupational cohorts exposed to asbestos has been lung cancer and mesothelioma, and we are not aware of any estimates of potency factors for NMRD in other cohorts. However, McDonald et al. (2004) reported a potency factor ($K_N = 0.0038$) for NMRD in the Libby cohort. This implies that the RR associated with a cumulative exposure of 100f/ml-y is approximately 1.38. This is slightly larger than the potency factor for lung cancer reported by McDonald et al. in the same paper. We analyzed NMRD in the Libby cohort using the Cox proportional hazards model and estimated $K_N = 0.0013$, slightly larger than the potency factor for lung cancer using the Cox model (see above). These modest potency factors do not suggest that there will be an epidemic of NMRD in the general population of Libby. This conclusion is supported by the ATSDR mortality study (ATSDR, 2000), which I discuss below, and which shows that mortality from NMRD is not increased in the general population of Libby, i.e. in the population not occupationally exposed to Libby asbestos.

To support his allegation that pleural disease from Libby asbestos exposure is "highly progressive", Dr. Whitehouse cites his fatally flawed 2004 study of lung function decrement in a cohort of his patients.

The Whitehouse Study

Many of Dr. Whitehouse's allegations regarding the toxicity of Libby fibers rely on a lung function study conducted by him. He (Whitehouse, 2004) claims, "[t]he progressive loss of pulmonary function in 76% of the 123 patients with pleural changes followed in this group of patients with Libby tremolite exposure is excessive compared to other published reports." The basis of this claim is a study published in 2004. There are a number of problems with the study that make interpretation of the reported results virtually impossible. The most serious problem with the study is that Dr. Whitehouse makes no attempt to relate decrements in lung function to the level of asbestos exposure in his study population. Therefore, no conclusions regarding the association of asbestos

exposure at Libby and loss of lung function can be drawn from this study. The other main problems with the study are the following.

1. The subjects of the study were not randomly chosen from the Libby population. The subjects were selected from among Dr. Whitehouse's patient population. It is highly likely that these subjects entered the study because of symptoms and therefore are not representative of the general Libby population. This problem of self-selection is compounded by the fact that of the 491 subjects originally in the study, the data on only 123 were used for analyses. Only the patients with repeated pulmonary function tests, who were likely the sickest, were included in this study. Because of this problem of selection, findings from the study cannot be generalized to the Libby population. Moreover, there was no control group.
2. Dr. Whitehouse does not use the most appropriate statistical methods to exploit the fact that individual level longitudinal data are available on each study subject. Since, at least on some patients, more than two sets of measurements were available, the appropriate way to analyze these data would be to consider each individual's response separately and address the correlations between consecutive readings by using standard statistical techniques. Instead, the method used by Dr. Whitehouse appears to use only the first and last sets of measurements and discards the others. Specifically, the whole problem should have been set up as a linear regression in which pulmonary capacity is modeled as a function of covariates such as age, gender, smoking history, obesity, radiographic changes, pleural thickening, and most importantly history of exposure to asbestos. The coefficients of such a model could be estimated using generalized estimating equations (GEE) techniques.
3. For his statistical method, Dr. Whitehouse has apparently used Analysis of Variance (ANOVA) with mean annual pulmonary function changes for each subject regressed against covariates. This method is not optimal for this data set because it results in some data being discarded. Moreover, Dr. Whitehouse uses this method incorrectly, which makes his results uninterpretable. First, he does not even present the actual regression equation that was used for his analyses. Second, he does not consider age, a potential strong predictor of pulmonary function, and other important covariates (see below) explicitly in his analyses. Instead he claims, "[s]ince the patient values were all age corrected against the normative predicted values, changes in the percentage predicted over time reflected changes of pulmonary function beyond that accounted for by aging." The 'normative predicted values' were apparently taken from a study done by Knudson and published in 1983. Dr. Whitehouse does not say how the effect of age can be adjusted using these normative values. Furthermore, normative values from a publication in 1983 can hardly be considered to be a contemporary gold standard for the effects of age on pulmonary functions. A study by Brodkin et al. (1996), which Dr. Whitehouse cites, used age as one of the covariates in the regression. It is, therefore, unclear why Dr. Whitehouse used values that were "age corrected against normative predicted values" instead of using age directly as a covariate.
4. Properly performed analyses would have allowed Dr. Whitehouse to relate lung function to various factors, such as age, cigarette smoking, obesity, radiographic

pleural changes measured on the ILO scale, pleural thickening, and most importantly, asbestos exposure, known to affect lung function. The paper by Brodkin et al. (1996) that he cites as a reference includes each of these covariates in analyses. In particular, Dr. Whitehouse makes no attempt to relate declines in pulmonary function to radiographic abnormalities or to levels of asbestos exposure at Libby. For example, among the study subjects are 86 former Grace employees, 27 family members of employees, and 10 individuals who were only environmentally exposed. If asbestos exposure in Libby were associated with declines in lung function, one would expect the largest declines in pulmonary function among the employees, followed by family members, with the smallest declines among those only environmentally exposed. From this study it is impossible to determine whether there were any decrements in lung function associated with asbestos exposure. The decrements observed in the study could be entirely attributable to other factors, such as age, smoking habits, and obesity. Whether individuals who were environmentally exposed to Libby amphiboles suffered any declines in lung function is one of the central questions. Dr. Whitehouse's study cannot address this question.

5. Dr. Whitehouse reports that he used a Sensormedics model 6200 to do the pulmonary function measurements before 1988 and a Medgraphics model 1085 since that time. His paper does not give any indication of the number of patients whose initial and final measurements were made on different machines and whether any attempt was made to cross-calibrate the machines.

Finally, there are apparent discrepancies and unclear statements on important issues in the Whitehouse paper. For example, he states, "[t]he parameters that were felt to be most valuable for analysis were forced vital capacity (FVC), (taking the best available and valid number from each set),..." Dr. Whitehouse does not explain how he chose the best available and valid number. Obviously, this choice could influence the results significantly. In another example of lack of clarity, figure 2 in the paper appears to show that the average FVC loss among 94 patients was 3.2% per year. However, the caption of figure 3 appears to say that only 79 patients had average FVC loss greater than 1% per year.

In summary, because of fundamental problems with the methodology, this study does not provide the information that would be required to determine to what extent loss of lung function among residents of Libby is attributable to their asbestos exposure.

Environmental Exposure to Libby Fibers and Disease

Dr. Whitehouse claims that his studies show that environmental (non-occupational) exposure to Libby asbestos poses a grave risk to the Libby population. The ATSDR studies, which investigated the health consequences of environmental exposure to Libby fibers, do not support this contention.

The Agency for Toxic Substances and Disease Registry (ATSDR) Studies

In response to concerns about asbestos exposure and its health consequences the ATSDR undertook two studies in the Libby area in 2000 and 2001. One was a mortality study, which used standard epidemiological procedures to investigate whether mortality from specific causes known to be associated with asbestos exposure was elevated in the Libby area. The second was a radiographic study of over 6,000 current or former residents of the Libby area. While historical occupational exposures to asbestos were high, the crucial issue here is if, and to what extent, environmental exposures contributed to the burden of asbestos-related disease in Libby. The ATSDR screening studies cannot address this issue as I discuss in more detail below. The mortality studies show that there was no increase in the risk of death from environmental exposure to Libby fibers.

The ATSDR Mortality Study

The ATSDR undertook a study of mortality from specific causes in the Libby area over the 20-year period 1978-1998. Numbers of deaths from specific causes were compared with numbers that would be expected under national and Montana death rates. Standard epidemiological and statistical techniques were used to compute Standardized Mortality Ratios (SMRs) and their confidence intervals. The causes of death of main interest were lung cancer, mesothelioma, and non-malignant respiratory disease (NMRD), particularly asbestosis. The ATSDR mortality study shows clearly that once deaths from lung cancer, mesothelioma and NMRD among the occupationally exposed are removed, the rates of these diseases are not increased in the Libby population. Thus the ATSDR study shows that there is no increase in mortality from lung cancer, mesothelioma or NMRD among those only environmentally exposed to Libby amphibole.

In its original report the ATSDR noted a small statistically non-significant increase in lung cancer deaths within Libby City and the extended Libby area using Montana death rates as the standard. With US death rates as the standard, no increase in lung cancer deaths was reported. This finding was surprising in view of the fact that SMRs were significantly increased in the occupational cohorts studied by Amandus and McDonald, and suggests that lung cancer may actually be decreased in residents of Libby who did not work at the mine. In an update to the original report, the ATSDR admitted to an incomplete mortality census in its original report and added 50 deaths from lung cancer to the original count of 80 in Central Lincoln County. With these additional deaths, the report indicates that lung cancer mortality in the Libby area was statistically significantly increased over expectation using both U.S. and Montana lung cancer death rates as the standards.

Inclusion of these 50 lung cancer deaths in the revised analyses does not appear to be scientifically defensible, and would be expected to inflate the true lung cancer mortality risk in Libby. Many of these lung cancer deaths apparently occurred in areas outside Libby and, in fact, outside the state of Montana. With no explanation, the ATSDR lists 11

states other than Montana (Arizona, Idaho, Kansas, Nebraska, New York, North Dakota, Oregon, Tennessee, Utah, Washington, and Wyoming) where these additional deaths among Libby residents could have occurred. It is not clear, however, how Libby residency was established for individuals who died outside the state of Montana. Some of these individuals could have gone to a neighboring state for medical treatment and died there. In this case, of course, counting these deaths as Libby deaths is legitimate. However, it is extremely unlikely that deaths occurring as far away as Tennessee and Nebraska, for example, were among individuals who had traveled there for treatment. Then the central issue is when these individuals had moved from Libby, even if they were considered Libby residents. If they had left the Libby area some years before death, then it is not legitimate to count these deaths as Libby deaths, unless some adjustment is made for this fact. For example, if these deaths are included, the 'person-year' calculation in the denominator should include all individuals who moved out of the Libby area during the period of the study, irrespective of disease status. Therefore, I believe that the ATSDR procedure very likely led to an over-count of the number of lung cancer deaths in the Libby area.

However, despite the inflated count of lung cancer in the ATSDR study, table 8 of the revised report indicates that, when former workers at the Libby plant were excluded, there was no increase in lung cancer deaths in the general population of the area. Thus, the number of lung cancer deaths over the period of the study offers no evidence that environmental exposures contributed to the lung cancer mortality in the area.

The ATSDR reports 3 cases of mesothelioma (there were 4 in the original study) among residents of Lincoln County over the period of the study. Since the background rate of mesothelioma is estimated to be approximately 2-4 per million individuals per year (Price & Ware, 2004; Moolgavkar et al., 2009), this number points to a statistically significant elevation of risk in the Libby area. An additional 8 mesothelioma deaths were identified among prior residents of Lincoln County. The updated McDonald study identifies a total of 12 mesothelioma deaths in the occupational cohort through 1998, suggesting that all 11 mesothelioma deaths reported by ATSDR were occupationally exposed. ATSDR reports, however, that at least one case had not had occupational exposure. Even if 1 mesothelioma death occurred in an individual not occupationally exposed, a single death could have occurred by chance in the Libby area over a 20 year period given the background rate of mesothelioma. In any event, I believe as in the case of lung cancer, this study offers no evidence that environmental exposure contributed to mesothelioma deaths in the Libby area. The latest update of the occupational cohort (Sullivan, 2007) reports 15 mesothelioma deaths, as noted above.

Among the causes of death other than cancer, of most interest are the non-malignant respiratory diseases (NMRD), particularly asbestosis. Eleven pneumoconiosis deaths among males and one among females are reported over the period of the study. All of these are labeled asbestosis in the ATSDR report, although it is not clear how this diagnosis was verified. However, as table 8 of the ATSDR report notes, only one of these occurs in an individual who was not occupationally exposed, with 11 occurring among occupationally exposed individuals. When these 11 deaths are removed, deaths from

pneumoconiosis were not elevated among those not occupationally exposed. There is no evidence that environmental exposures contributed to the deaths from pneumoconiosis.

The ATSDR reports also that deaths from non-malignant respiratory causes other than pneumoconiosis were statistically significantly elevated in the Libby area. There were 14 NMRD deaths among males and 4 among females. Again, table 8 indicates that the increase in deaths from this cause was limited to those occupationally exposed.

In conclusion, there is no evidence that environmental exposure to asbestos contributed to the deaths from lung cancer, mesothelioma and NMRD, including asbestosis, in the Libby area.

The ATSDR Medical Testing Study

The ATSDR issued a report in August 2001 on the medical testing of individuals in the Libby area. The bulk of the report deals with the findings of a radiographic study on volunteers who had lived in the Libby area for at least six months prior to 1990. The study, which has been published in the peer-reviewed literature (Peipins et al., 2003), is not a proper epidemiological study because the participants were self-selected and there was no control group. The ATSDR (2001) acknowledged as much, saying, "[t]he study was not designed as an analytic epidemiologic study with comparison groups."[5] Multivariate logistic regression analyses were conducted to examine the association of covariates such as age, gender, smoking habits, body mass index (BMI) and, most importantly, exposure to asbestos. A number of covariates were found to be significantly associated with radiographic abnormalities including age, gender, and smoking habits. With respect to asbestos exposure, activities that could have led to significant exposures were also found to be significantly associated with radiographic abnormalities. Thus the strongest risk factor for radiographic abnormalities was being a former WRG worker. 'Recreational' exposures to asbestos that could have resulted from activities such as playing in vermiculite piles were also associated with radiographic changes.

This study has significant limitations that preclude any conclusions regarding associations between environmental exposure to Libby amphibole and abnormalities on chest x-rays. The main limitations are the following.

1. The study group was self-selected with all the attendant biases of such self-selection. In particular, the study subjects cannot be considered to be representative of the general population of the Libby area.
2. The x-ray readers were not blinded to the source of the plates, and no control plates were used. That is, the readers were aware that the plates were from residents of Libby and that the focus of the investigation was asbestos associated

[5]The ATSDR has repeatedly emphasized the fact that the screening study was not designed as an epidemiological study. In addition to the statement from the report quoted here, the ATSDR made similar statements in their report to the community on preliminary findings (February 2001) and in a briefing to the Montana Congressional and Gubernatorial Staffs (e-mail from Jason E. Broehm to Jeffrey Lybarger et al., dated Aug 23, 2001, subject: draft Libby congressional briefing summary for comment.).

radiographic abnormalities. In a properly designed epidemiological study, control plates from other locations would have been included, and the readers would have been blinded to the source of the plates they were reading.

3. There was no control group so that a background rate for radiographic changes could not be established. In a properly designed epidemiological study a control group would have been included. In the absence of a control group, it is impossible to assess whether the frequency of radiographic abnormalities observed in those environmentally exposed is elevated. In response to a letter by Price (2004), the authors contend, "[t]he rate of 6.7% for the no-apparent-exposure group in our analysis…is considerably higher than the prevalence rates of pleural abnormalities found in published studies of other nonoccupationally exposed populations in the United States, which range from 0.2% among blue-collar workers in North Carolina (Castellan et al., 1985) to 2.3% among patients Veterans Affairs hospitals in New Jersey (Miller and Zurlo, 1996)." With the rapid advances in medical imaging technology, to compare the results of the ATSDR study with one conducted in 1985 (Castellan) is disingenuous at best. The design of the second study by Miller and Zurlo cited by Peipins et al. is different in so many ways from the ATSDR study that any comparison of results is meaningless.

In summary, because the ATSDR radiographic study was not designed to be an epidemiological study, it is, at best, a hypothesis generating study that needs to be followed up with a properly designed epidemiological study.

The radiographic studies conducted by Amandus et al (1987) and McDonald et al (1986) are relevant to this discussion. The results of these studies clearly indicate that, when radiographic end-points are considered, there is no evidence to suggest that Libby fibers are any more toxic than other asbestos fibers. In a later study, McDonald et al. (1988) found no increase in radiographic abnormalities over controls in a cohort of vermiculite miners exposed to small amounts of fibrous tremolite.

Conclusions

Any risk assessment at Libby should follow well established quantitative principles. Whether an individual comes into contact with expanded vermiculite product or vermiculite concentrate, ultimately the risk depends on the level of exposure to Libby asbestos. A number of studies of the occupational cohort at Libby allow the estimation of potency factors of Libby asbestos for lung cancer, mesothelioma, and NMRD. These potency factors can be used to assess the risks for the general population at Libby given exposure information. In particular, given exposure information, it is possible to predict the number of deaths from these diseases that would be expected to occur in the Libby population in the future. Since the potency factors described here are specific to Libby fibers, these projections would be expected to be more accurate than those based on the generic potency factors for asbestos developed by the EPA in 1986, which would overestimate the number of deaths. The potency factors for Libby fibers indicate also that

Libby fibers are considerably less toxic than crocidolite. The toxicity of Libby fibers appears to lie somewhere between the toxicity of chrysotile and that of amosite.

Suresh H. Moolgavkar, M.D., Ph.D.

Bellevue, WA 98004
April 6, 2009

Suresh Moolgavkar, M.D., Ph.D. Compensation

I am being compensated for this report at $550 per hour.

References

Amandus HE, Wheeler R. 1987. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite part II. Mortality. Am J Ind Med 11:15-26.

ATSDR. 2001. 2000 medical testing of individuals potentially exposed to asbestiform minerals associated with vermiculite in Libby, Montana. A report to the community. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2002. Mortality in Libby, Montana, 1979-1998. Libby Asbestos Site, Libby, Lincoln County, Montana. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

Berman DW, Crump KS. 2008. Update of Potency Factors for Asbestos-Related Lung Cancer and Mesothelioma. Crit Rev Toxicol 38 (Suppl 1):1-47.

Brodkin CA, Barnhart S, Checkoway H, Balmes J, Omenn GS and Rosenstock L. 1996. Longitudinal pattern of reported respiratory symptoms and accelerated ventilatory loss in asbestos-exposed workers. Chest 109:120-126.

Hodgson JT and Darnton A. 2000. The quantitative risk of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occ Hyg 44:565-601.

Larson T and Phelan M. Survival analysis of a cohort of workers exposed to amphibole-contaminated vermiculite. Agency for Toxic Substances and Disease Registry. Unpublished manuscript.

Loomis D, Dement JM, Wolf SH, Richardson DB. 2009. Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers. E-published.

McDonald JC, McDonald AD, Armstrong B, Sebastien P. 1986. Cohort study of vermiculite miners exposed to tremolite. Br J Ind Med 43:436-444.

McDonald JC, McDonald AD, Sebastian P, Moy K. 1988. Health of vermiculite miners exposed to trace amount of fibrous tremolite. Br J Ind Med 43:630-634.

McDonald JC, Harris J, Armstrong B. 2004. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. Occup Environ Med 61:363--366.

Moolgavkar SH. June 11, 2007. W.R. Grace & Co. Bankruptcy supplemental report on asbestos and disease causation.

Moolgavkar SH. July 31, 2007. W. R. Grace & Co. Bankruptcy supplemental/rebuttal report on asbestos and disease causation.

Moolgavkar SH, Meza R, Turim J. 2009. Pleural and peritoneal mesotheliomas in SEER: age effects and temporal trends, 1973-2005. Can Cause Control. E-published.

Peto J. 1979. Dose-response relationships for asbestos-related disease, implications for hygiene standards part II. Mortality. Ann NY Acad Sci 330:195-203.

Peipins LA, Lewin M, Campolucci, Lybarger JA, Miller A, Middleton D, Weis C, Spence M, Black B, Kapil V. Radiographic abnormalities and exposure to asbestos-contaminated vermiculite in the community of Libby, Montana, USA. Environ Health Perspect 111:1753-1759. 2 Am J Ind Med 003.

Price B and Ware A. 2004. Mesothelioma trends in the United States: an update. Am J Epidemiol 159:107-112.

Sullivan PA. 2007. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect 115:579-585.

Whitehouse AC. 2004. Asbestos-related pleural disease due to tremolite associated with progressive loss of lung function. Am J Ind Med 46:219–225.

Whitehouse AC, Black CB, Heppe MS, Ruckdeschel J, Levin SM. 2008. Environmental exposure to Libby asbestos and mesotheliomas. Am J Ind Med 51:877-880.

Exponent

Exponent
15375 SE 30th Place
Suite 250
Bellevue, WA 98007

telephone 425-519-8700
facsimile 425-519-8799
www.exponent.com

Suresh H. Moolgavkar, M.D., Ph.D.
Corporate Vice President and Director of the Center for Epidemiology, Biostatistics, and Computational Biology

Professional Profile

Dr. Suresh Moolgavkar has more than 30 years of experience in the fields of epidemiology, biostatistics, and quantitative risk assessment. He is internationally known for his work in developing mechanistically based dose-response models for carcinogenesis, and, in particular, for the two-mutation clonal expansion model, also known as the Moolgavkar-Venzon-Knudson (MVK) model. For the past decade, Dr. Moolgavkar has also been keenly interested in air pollution epidemiology. Dr. Moolgavkar retired from his position as a Full Member of the Fred Hutchinson Cancer Research Center in August 2008. He continues to be an Affiliate Investigator at the Center and Professor of Epidemiology and Adjunct Professor of Biostatistics and Applied Mathematics at the University of Washington in Seattle. Dr. Moolgavkar has served on the faculties of Johns Hopkins University, Indiana University, University of Pennsylvania, and Fox Chase Cancer Center. He has been a visiting scientist at the Radiation Effects Research Foundation in Hiroshima, the International Agency for Research on Cancer in Lyon, and the German Cancer Research Center in Heidelberg. Dr. Moolgavkar has served on numerous review panels and as a consultant to the National Cancer Institute, EPA, Health and Welfare, Canada, The International Agency for Research on Cancer, the California Air Resources Board, and the CIIT Centers for Health Research, among others.

Dr. Moolgavkar is the author or co-author of more than 150 papers and contributed chapters in the areas of epidemiology, biostatistics, and quantitative risk assessment, and has edited three books in these areas. He was the senior editor of a monograph, *Quantitative Estimation and Prediction of Human Cancer Risk*, published by the International Agency for Research on Cancer. He is an elected member of the American Epidemiological Society. Dr. Moolgavkar has served on the editorial board of *Genetic Epidemiology*, is currently Associate Editor for Health and Environment of *Risk Analysis—An International Journal*, and on the editorial board of *Inhalation Toxicology*.

Dr. Moolgavkar's research has been supported largely by grants from the National Institutes of Health, the U.S. Department of Energy, and EPA.

Academic Credentials and Professional Honors

Senior Fellow, Department of Epidemiology, University of Washington, 1976–1977
Ph.D., Mathematics, Johns Hopkins University, Baltimore, Maryland, 1973
Postdoctoral Fellow, Departments of Pharmacology and Biophysics, Johns Hopkins Medical School, Baltimore, Maryland, 1966–1968
M.B.B.S., (M.D.) Bombay University, 1965

Elected Member, American Epidemiological Society
Distinguished Achievement Award, Society for Risk Analysis, 2001
Founders' Award, Chemical Industry Institute of Toxicology, 1990
Lester R. Ford Award of Mathematical Association of America, 1977
Faculty Research Fellowship of Indiana University, 1974–1976

Academic Appointments

Professor, Department of Epidemiology, University of Washington, 1984–present
Adjunct Professor, Department of Biostatistics, University of Washington, 1984–present
Adjunct Professor, Department of Applied Mathematics, University of Washington, 2004–present
Member, The Fred Hutchinson Cancer Research Center, Seattle, 1984–2008
Affiliate Investigator, The Fred Hutchinson Cancer Research Center, 2008–present
Member, Graduate Faculty, University of Washington, 1984–present

Academic Appointments

Adjunct Professor, Department of Applied Mathematics, University of Washington, 2004–present
Adjunct Professor, Department of Biostatistics, University of Washington, 1984–present
Member, The Fred Hutchinson Cancer Research Center, Seattle, 1984–present
Member, Graduate Faculty, University of Washington, 1984–present
Professor, Department of Epidemiology, University of Washington, 1984–present
Adjunct Associate Professor, Department of Research Medicine, University of Pennsylvania School of Medicine, 1980–1984
Research Physician, The Institute for Cancer Research, Fox Chase Cancer Center, Philadelphia, 1979–1984
Clinical Assistant Professor, Department of Research Medicine, University of Pennsylvania School of Medicine, 1977–1980
Associate, American Oncologic Hospital, Philadelphia, 1977–1984
Epidemiologist, The Fox Chase Cancer Center, Philadelphia, 1977–1984
Member, Graduate Group in Epidemiology, University of Pennsylvania, 1977–1984
Assistant Professor of Mathematics, Indiana University, Bloomington, 1973–1977
Instructor in Mathematics, Johns Hopkins University, 1972–1973

Editorships and Editorial Review Boards

Editorial Board, *Inhalation Toxicology*, 2006–present
Guest Editor, *Modeling and Data Analysis in Cancer Studies*, special issue of Mathematical and Computer Modelling, 33(12–13), 2001
Associate Editor, *Risk Analysis—An International Journal*, 2000–present
Senior Editor, *Quantitative Estimation and Prediction of Human Cancer Risk*, International Agency for Research on Cancer, Scientific Publications 131, 1999
Editor, *Scientific Issues in Quantitative Cancer Risk Assessment*, Birkhauser, Boston, 1990
Co-Editor, *Modern Statistical Methods in Chronic Disease Epidemiology*, John Wiley, 1986
Editorial Board, *Genetic Epidemiology*, 1984–1988

Publications

Mathematical

Ewing J, Moolgavkar S, Smith L, Stong RE. Stable parallelizability of lens spaces. J Pure Appl Algebra 1977; 10:177–191.

Ewing J, Moolgavkar S. Euler characteristics of complete intersections. Proc Am Math Soc 1976; 56:390–391.

Ewing J, Gustafson E, Halmos P, Moolgavkar S, Wheeler W, Ziemer W. American mathematics from 1940 to the day before yesterday. Am Math Monthly 1976; 83:503–516.

Ewing J, Moolgavkar S. On a conjecture of Atiyah and Thom. Preprint, Indiana University, 1976.

Ewing J, Moolgavkar S. On the group of holomorphic line bundles on an algebraic surface. Preprint, Indiana University, 1976.

Moolgavkar S. On the existence of a universal germ of deformations for elliptic pseudo group structures on compact manifolds. Trans Am Math Soc 1975; 212:173–197.

Ewing J, Moolgavkar S. On the signature of Fermat surfaces. Michigan Math J 1975; 22:257–268.

Biomedical

Meza R, Jeon J, Moolgavkar SH, Luebeck EG. The age-specific incidence of cancer: Phases, transitions and biological implications. PNAS, in press.

Luebeck EG, Moolgavkar SH, Liu A, Ulrich N. Does folic acid supplementation prevent or promote colon cancer? Results from model-based predictions. Cancer Epidemiol Biomarkers Prevent 2008; 17:1360–1367.

Little M, Heidenreich W, Moolgavkar SH, Schoellnberger H, Thomas DC. Systems biological and mechanistic modelling of radiation-induced cancer. Rad Environ Biophys 2008; 47:39–47.

Meza R, Hazelton WD, Colditz GA, Moolgavkar SH. Analysis of lung cancer incidence in the nurses' health and the health professionals' follow-up studies using a multistage carcinogenesis model. Cancer Causes Control 2008; 19:317–328.

Jeon J, Meza R, Moolgavkar SH, Luebeck EG. The evaluation of cancer screening strategies using multistage carcinogenesis models. Math Biosci 2008; 213:56–70.

Reiss R, Anderson EL, Cross CE, Hidy G, Hoel D, McClellan R, Moolgavkar S. Evidence of health impacts of sulfate and nitrate containing particles in ambient air. Inhal Toxicol 2007; 19:419–449.

Moolgavkar SH. Pollution analysis flawed by statistical model. Correspondence. Nature 2007; 445:21.

Hazelton WD, Moolgavkar SH, Curtis SB, Zielinski JM, Ashmore JP, Krewski D. Biologically based analysis of lung cancer incidence in a large Canadian occupational cohort with low-dose ionizing radiation exposure, and comparison with Japanese atomic bomb survivors. J Toxicol Environ Health 2006; 69:1013–1038.

Moolgavkar SH. Fine particles and mortality. Inhal Toxicol 2006; 18:93–94.

Jeon J, Luebeck EG, Moolgavkar SH. Age effects and temporal trends in adenocarcinoma of esophagus and gastric cardia. Cancer Causes Control 2006; 17:971–981.

Clements MS, Hakulinen T, Moolgavkar SH. Bayesian projections: What are the effects of excluding data from the younger age groups? Am J Epidemiol 2006; 164:292–293.

Luebeck EG, Moolgavkar SH. Biological and mathematical aspects of multistage carcinogenesis. In: Quantitative Methods for Cancer and Human Health Risk Assessment. Edler I, Kitsos CP (eds), Wiley-Liss, 2005.

Luebeck EG, Buchmann A, Schneider D, Moolgavkar SH, Schwarz M. Modulation of liver tumorigenesis in Connexin32-deficient mouse. Mutat Res 2005; 570:33–47.

Moolgavkar SH. A review and critique of the EPA's rationale for a fine particle standard. Regulat Toxicol Pharmacol 2005; 42:123–144.

Hazelton WD, Clements MS, Moolgavkar SH. Multistage carcinogenesis and lung cancer mortality in three cohorts. Cancer Epidemiol Biomarkers Prevent 2005; 14:1171–1181.

Clements MS, Armstrong B, Moolgavkar SH. Lung cancer rate predictions using generalized additive models. Biostatistics 2005; 6:576–589.

Dewanji A, Luebeck EG, Moolgavkar SH. A generalized Luria-Delbruck process. Math Biosci 2005; 197:140–152.

Meza R, Luebeck EG, Moolgavkar SH. Gestational mutations and carcinogenesis. Math Biosci 2005; 197:188–210.

Zheng CJ, Luebeck EG, Byers B, Moolgavkar SH. On the number of founding germ cells in humans. Theor Biol Med Model 2005; 24:2, 32.

Curtis SB, Hazelton WD, Luebeck EG, Moolgavkar SH. From mechanism to risk estimation—bridging the chasm. Adv Space Res 2004; 34:1404–1409.

Moolgavkar SH. Fifty years of the multistage model: Remarks on a landmark paper. Int J Epidemiol 2004; 33:1182–1183.

Little MP, Blettner M, Boice JD Jr, Bridges BA, Cardis E, Charles MW, de Vathaire F, Doll R, Fujimoto K, Goodhead D, Grosche B, Hall P, Heidenreich WF, Jacob P, Moolgavkar SH, Muirhead CR, Niwa O, Paretzke HG, Richardson RB, Samet JM, Sasaki Y, Shore RE, Straume T, Wakeford R. Potential funding crisis for the Radiation Effects Research Foundation. Lancet 2004; 364:557–558.

Heidenreich WF, Luebeck EG, Hazelton WD, Paretzke HG, Moolgavkar SH. Response to the commentary of Donald A. Pierce (Radiat Res 2003; 160:718–723). Radiat Res 2004; 161:369–370.

Heidenreich WF, Luebeck EG, Moolgavkar SH. Effects of exposure uncertainties in the TSCE model and application to the Colorado miners data. Radiat Res 2004; 161:72–81.

Moolgavkar SH, Luebeck EG. Multistage carcinogenesis and the incidence of human cancer. Genes Chromosomes Cancer 2003; 38:302–306.

Moolgavkar SH. Air pollution and daily mortality in two U.S. counties: season-specific analyses and exposure-response relationships. Inhal Toxicol 2003; 15:877–907.

Moolgavkar SH. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook counties. pp. 183–198. In: Health Effects Institute Special Report, Revised Analyses of Time-Series Studies of Air Pollution and Health. Health Effects Institute, 2003.

Krewski D, Zielinski JM, Hazelton WD, Garner MJ, Moolgavkar SH. The use of biologically based cancer risk models in radiation epidemiology. Radiat Prot Dosimetry 2003; 104:367–76.

Gregori G, Hanin L, Luebeck G, Moolgavkar S, Yakovlev A. Testing goodness of fit for stochastic models of carcinogenesis. Math Biosci 2002; 175:13–29.

Heidenreich WF, Luebeck EG, Hazelton WD, Paretzke HG, Moolgavkar SH. Multistage models and the incidence of cancer in the cohort of A-bomb survivors. Rad Res 2002; 158:607–614.

Dewanji A, Moolgavkar SH. Choice of stratification in Poisson process analysis of recurrent event data with environmental covariates. Statist Med 2002; 21:3383–3393.

Curtis SB, Luebeck EG, Hazelton WD, Moolgavkar SH. Does radiation enhance promotion of already-initiated cells via a bystander effect? Int Congress Series 2002; 1236:283–287.

Curtis SB, Luebeck EG, Hazelton WD, Moolgavkar SH. A new perspective of carcinogenesis from protracted high-LET radiation arises from the two-stage clonal expansion model. Adv Space Res 2002; 30:937–944.

Luebeck EG, Moolgavkar SH. Multistage carcinogenesis and the incidence of colorectal cancer. Proc National Acad Sci 2002; 99:15095–15100.2.

Moolgavkar SH, Turim J, Brown RC, Luebeck EG. Long man-made fibers and lung cancer risk. Regulat Toxicol Pharmacol 2001; 33:138–146.

Hazelton WD, Luebeck EG, Heidenreich WF, Moolgavkar SH. Analysis of a historical cohort of Chinese tin miners with arsenic, radon, cigarette, and pipe smoke exposures using the biologically-based two-stage clonal expansion model. Rad Res 2001; 156:7–94.

Moolgavkar SH, Turim J, Brown RC. The power of the European Union protocol to test for carcinogenicity of inhaled fibers. Regulat Toxicol Pharmacol 2001; 33:350–355.

Moolgavkar SH, Brown RC, Turim J. Biopersistence, fiber length, and cancer risk assessment for inhaled fibers. Inhal Toxicol 2001; 13:755–772.

Moolgavkar SH, Luebeck EG, Turim J, Brown RC. Lung cancer risk associated with exposure to man-made fibers. Drug Chem Toxicol 2000; 23:223–242.

Moolgavkar SH, Hazelton WF, Luebeck EG, Levy D, Sheppard L. Air pollution, pollens, and admissions for chronic respiratory disease in King County. Inhal Toxicol 2000; 12(Supplement 1):157–171.

Dewanji A, Moolgavkar SH. A Poisson process approach for recurrent event data with environmental covariates. Environmetrics 2000; 11:665–673.

Moolgavkar SH. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. J. Air Waste Manage Assoc 2000; 50:1199–1206.

Moolgavkar SH. Air pollution and daily mortality in three U.S. counties. Environ Health Perspect 2000; 108:777–784.

Moolgavkar SH. Air pollution and hospital admissions for chronic obstructive pulmonary disease in three metropolitan areas in the US. Inhal Toxicol 2000; 12(Suppl 4):75–90.

Luebeck EG, Buchmann A, Stinchcombe S, Moolgavkar SH, Schwarz M. Effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) on initiation and promotion of GSTP-positive foci in rat liver: A quantitative analysis of experimental data using a stochastic model. Toxicol Appl Pharmacol 2000; 167:63–73.

Grasl-Kraupp B, Luebeck G, Wagner A, Loew-Baselli A, De Gunst M, Waldhor T, Moolgavkar S, Schulte-Hermann R. Quantitative analysis of tumor initiation in rat liver: Role of cell replication and cell death (apoptosis). Carcinogenesis 2000; 21:1411–1421.

Moolgavkar SH, Moller H, Woodward A. Principles of the epidemiologic approach to quantitative estimation and prediction of cancer risk. pp. 61–74. In: Quantitative Estimation and Prediction of Cancer Risk. Moolgavkar SH, Krewski D, Zeise L, Cardis E, Moller H (eds), IARC Scientific Publications, 1999.

Moolgavkar SH, Krewski D, Schwarz M. Mechanisms of carcinogenesis and biologically-based models for quantitative estimation and prediction of cancer risk. pp. 179–238. In: Quantitative Estimation and Prediction of Cancer Risk. Moolgavkar SH, Krewski D, Zeise L, Cardis E, Moller H (eds), IARC Scientific Publications, 1999.

Moolgavkar SH, Woodward A, Krewski D, Cardis E, Zeise L. Future perspectives and research needs. pp. 305–322. In: Quantitative Estimation and Prediction of Cancer Risk. Moolgavkar SH, Krewski D, Zeise L, Cardis E, Moller H (eds), IARC Scientific Publications, 1999.

Cardis E, Zeise L, Schwarz M, Moolgavkar S. Review of specific examples of QEP. pp. 239–304. In: Quantitative Estimation and Prediction of Cancer Risk. Moolgavkar SH, Krewski D, Zeise L, Cardis E, Moller H (eds), IARC Scientific Publications, 1999.

Moolgavkar SH, Luebeck EG, Turim J, Hanna L. Quantitative assessment of the risk of lung cancer associated with occupational exposure to refractory ceramic fibers. Risk Anal 1999; 19:599–611.

Dewanji A, Goddard M, Krewski D, Moolgavkar SH. Two stage model for carcinogenesis: Number and size distributions of premalignant clones in longitudinal studies. Math Biosci 1999; 155:1–12.

Luebeck EG, Heidenreich WF, Hazelton WD, Paretzke HG, Moolgavkar SH. Biologically-based analysis of the data for the Colorado Plateau uranium miners cohort: Age, dose and dose-rate effects. Rad Res 1999; 152:339–351.

Moolgavkar SH. Stochastic models for estimation and prediction of cancer risk. pp. 237–259. In: Statistics for the Environment 4: Pollution Assessment and Control. Barnett V, Stein A, Feridun Turkman K (eds), John Wiley, NY, 1999.

Moolgavkar SH, Luebeck EG, Anderson EL. Estimation of unit risk for coke oven emissions. Risk Anal 1998; 18:813–825.

Gaylor DW, Moolgavkar S, Krewski D, Goldstein LS. Recent bioassay results on coal tars and benzo[a]pyrene: Implications for risk assessment. Regul Toxicol Pharmacol 1998; 28:178–179.

Moolgavkar SH. Comments on papers on U-shaped dose-response relationships for carcinogens. Hum Exper Toxicol 1998; 17:708–710.

Moolgavkar SH. Two-mutation carcinogenesis model. pp. 4635–4639. In: Encyclopedia of Biostatistics. Armitage P, Colton T (eds), John Wiley, 1998.

Moolgavkar SH, Lee JAH, Stevens RG. Analysis of vital statistical data. In: Modern Epidemiology. 2nd edition. Rothman K, Greenland S (eds), Lippincott-Raven, PA, 1998.

Moolgavkar SH, Luebeck EG, Anderson, EL. Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. Epidemiology 1997; 8(4):364–370.

Heidenreich W, Luebeck EG, Moolgavkar SH. Some properties of the hazard function of the two-mutation clonal expansion model. Risk Anal 1997; 17:391–399.

Moolgavkar SH. Stochastic cancer models: application to analyses of solid cancer incidence in the cohort of A-bomb survivors. Nucl Ener 1997; 36(6):447–451.

Kai M, Luebeck EG, Moolgavkar SH. Analysis of solid cancer incidence among atomic bomb survivors using a two-stage model of carcinogenesis. Rad Res 1997; 148:348–358.

Luebeck EG, Moolgavkar SH. Biologically based cancer modelling. Drug Chem Toxicol 1996; 19:221–243.

Luebeck EG, Curtis SB, Cross FT, Moolgavkar SH. Two-stage model of radon-induced malignant lung tumors in rats: effects of cell killing. Rad Res 1996; 145:163–173.

Moolgavkar SH, Luebeck EG, Buchmann A, Bock KW. Quantitative analysis of enzyme-altered foci in rats initiated with diecthylnitrosamine and promoted with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin or 1,2,3,4,6,7,8-heptachloro-*p*-dioxin. Toxicol Appl Pharmacol 1996; 138:31–42.

Moolgavkar SH, Luebeck EG, Hall TA, Anderson EL. Particulate air pollution and mortality. Letter to the Editor. Epidemiology 1996; 7:212–213.

Moolgavkar SH, Luebeck EG. A critical review of the evidence on particulate air pollution and mortality. Epidemiology 1996; 7:420–428.

Leroux BG, Lesenring WM, Moolgavkar SH, Faustman EM. A biologically based dose-response model for developmental toxicology. Risk Anal 1996; 16:449–458.

Dewanji A, Luebeck EG, Moolgavkar SH. A biologically-based model for the analysis of premalignant foci of arbitrary shape. Math Biosci 1996; 135:55–68.

Moolgavkar SH, Luebeck EG, Hall TA, Anderson EL. Particulate air pollution, sulfur dioxide, and daily mortality: A reanalysis of the Steubenville data. Inhal Toxicol 1995; 7:35–44.

Schwarz M, Buchmann A, Stinchcombe S, Luebeck EG, Moolgavkar SH, Bock KW. Role of receptors in human and rodent hepatocarcinogenesis. Mutat Res 1995.

Luebeck EG, Grasl-Kraupp B, Timmermann-Trosiener I, Bursch W, Schulte-Hermann R, Moolgavkar SH. Growth kinetics of enzyme altered liver foci in rats treated with phenobarbital or α-hexachlorocyclohexane. Toxicol Appl Pharmacol 1995; 130:30–315.

Luebeck EG, Moolgavkar SH. Biologically based cancer modeling. pp. 533–555. In: Toxicology and Risk Assessment. Fan AM, Chang LW (eds), Marcel Dekker, Inc., New York, 1995.

Moolgavkar SH. When and how to combine results from multiple epidemiological studies in risk assessment. pp. 77–90. In: The Proper Role of Epidemiology in Regulatory Risk Assessment. Graham J (ed), Elsevier, New York, 1995.

Moolgavkar SH, Luebeck EG, Hall TA, Anderson EL. Air pollution and daily mortality in Philadelphia. Epidemiology 1995; 6:476–484.

Moolgavkar SH, Luebeck EG. Incorporating cell proliferation kinetics into models for cancer risk assessment. Toxicology 1995; 102:141–147.

Stayner L, Smith R, Bailer J, Luebeck EG, Moolgavkar SH. Methods for modelling occupational studies for cancer risk assessment. Am J Indust Med 1995; 27:155–170.

Luebeck EG, Moolgavkar SH. Simulating the process of carcinogenesis. Math Biosci 1994; 123:127–146.

Moolgavkar SH. Air pollution and mortality (letter). N Eng J Med 1994; 330:1237–1238.

Moolgavkar SH. Biological models of carcinogenesis and quantitative cancer risk assessment. Guest Editorial. Risk Anal 1994; 14:879–882.

Moolgavkar SH. Cell proliferation and carcinogenesis models: General principles with illustrations from the rodent liver system. Environ Health Perspect 1993; 101(Suppl. 5):91–94.

Moolgavkar SH, Luebeck EG, Krewski D, Zielinski JM. Radon, cigarette smoke, and lung cancer: A reanalysis of the Colorado Plateau miners' data. Epidemiology 1993; 4:204–217.

Moolgavkar SH, Luebeck EG. A two-mutation model for radiation carcinogenesis in humans and rodents. pp. 199–210. In: New Frontiers in Cancer Causation. Iversen OH (ed), Taylor and Francis, Washington, DC, 1993.

Zheng CJ, Byers B, Moolgavkar SH. Allelic instability in mitosis: A unified model for dominant disorders. Proc Natl Acad Sci 1993; 90:10178–10182.

Moolgavkar SH, Luebeck EG. Interpretation of labelling indices in the presence of cell death. Carcinogenesis 1992; 13:1007–1010.

Moolgavkar SH, Luebeck EG. Risk assessment of non-genotoxic carcinogens. Toxicol Lett 1992; 64/65:631–636.

Moolgavkar SH. A population perspective on multistage carcinogenesis. pp. 381–392. In: Multistage Carcinogenesis. Proc. 22nd International Symposium of The Princess Takamatsu

Cancer Research Fund. Harris CC, Hirohashi S, Ito N, Pitot HC, Sugimura T, Terada M Yokota J (eds), Japan Scientific Societies Press, Tokyo, 1992.

Moolgavkar SH. Cancer models. pp. 239–252. In: Biophysical Modelling of Radiation Effects. Chadwick K, Moschini G, Varma M (eds), Adam Hilger, Bristol, 1992.

Moolgavkar SH. Carcinogenesis models: An overview. pp. 767–781. In: Indoor Radon and Lung Cancer: Reality or Myth? Cross FT (ed), Battelle Press, 1992.

Luebeck EG, Moolgavkar SH. Stochastic analysis of intermediate lesions in carcinogensis experiments. Risk Anal 1991; 11:149–157.

Dewanji A, Moolgavkar SH, Luebeck EG. Two-mutation model for carcinogenesis: Joint analysis of premalignant and malignant lesions. Math Biosci 1991; 104:97–109.

Nandakumar A, Davis S, Moolgavkar S, Witherspoon R, Schwartz S. Myeloid leukemia following therapy for a first primary cancer. Br J Cancer 1991; 63:782–788.

Moolgavkar SH. Cell proliferation in carcinogenesis (letter). Science 1991; 251:143.

Moolgavkar SH, Luebeck EG. The role of somatic mutations and cell replication kinetics in quantitative cancer risk assessment. pp. 469–479. In: Chemically Induced Cell Proliferation: Implications for Risk Assessment. Butterworth BE, Slaga TJ, Farland W, McClain M (eds), Wiley Liss, 1991.

Luebeck EG, Moolgavkar SH, Buchman A, Schwarz M. Effects of polychlorinated biphenyls in rat liver: Quantitative analysis of enzyme altered foci. Toxicol Appl Pharmacol 1991; 111:469–484.

Moolgavkar SH, Luebeck EG. Multistage carcinogenesis: A population-based model for colon cancer. JNCI 1991; 84:610–618.

Luebeck EG, Moolgavkar SH. Stochastic description of initiation and promotion in experimental carcinogenesis. Annali dell'Istituto Superiore di Sanita 1991; 27: 575–580.

Moolgavkar SH. Stochastic models of carcinogenesis. pp. 373–393. In: Handbook of Statistics, Volume 8. Rao CR, Chakraborty R (eds), Elsevier, 1991.

Moolgavkar SH, Cross FT, Luebeck G, Dagle GE. A two-mutation model for radon-induced lung tumors in rats. Rad Res 1990; 121:28–37.

Moolgavkar SH, Luebeck G. Two-event model for carcinogenesis: Biological, mathematical and statistical considerations. Risk Anal 1990; 10:323–341.

Moolgavkar SH, Luebeck G, DeGunst M. Two mutation model for carcinogenesis: Relative roles of somatic mutations and cell proliferation in determining risk. pp. 136–152. In:

Scientific Issues in Quantitative Cancer Risk Assessment. Moolgavkar SH (ed), Birkhauser, Boston, 1990.

Moolgavkar SH, Luebeck G., de Gunst M, Port RE, Schwarz M. Quantitative analysis of enzyme altered foci in rat hepatocarcinogenesis experiments. Carcinogenesis 1990; 11:1271–1278.

Moolgavkar SH. Cancer models, invited editorial. Epidemiology 1990; 1:419–420.

Dewanji A, Venzon DJ, Moolgavkar SH. A stochastic two-stage model for cancer risk assessment II: The number and size of premalignant clones. Risk Anal 1989; 9:179–186.

Moolgavkar SH. Multistage models for cancer risk assessment. pp. 9–20. In: Biologically Based Methods for Cancer Risk Assessment. Travis C (ed), NATO ASI Series A: Life Science Vol. 159, Plenum NY, 1989.

Moolgavkar SH, Dewanji A, Luebeck G. Cigarette smoking and lung cancer: A reanalysis of the British doctors' data. JNCI 1989; 81:415–420.

Moolgavkar SH. Dominant inheritance of colonic polyps and adenocarcinomas. N Engl J Med 1989; 320:316.

Hahn RA, Moolgavkar SH. Nulliparity, decade of first birth and breast cancer in Connecticut cohorts. Am. J. Public Health 1989; 79:1503–1507.

Moolgavkar SH. A two-stage carcinogenesis model for risk assessment. Cell Biol Toxicol 1989; 5:445–460.

Moolgavkar SH, Dewanji A. Combined effect of childbearing, menstrual events, and body size on age-specific breast cancer risk. Am J Epidemiol 1988; 128:1177–1178.

Venzon DJ, Moolgavkar SH. Origin invariant relative risk functions for case-control and survival studies. Biometrika 1988; 75:325–333.

Venzon DJ, Moolgavkar SH. An algorithm for computing profile-likelihood-based confidence intervals. Appl Stat 1988; 37:87–94.

Moolgavkar SH, Dewanji A. Biologically-based models for cancer risk assessment: A cautionary note. Risk Anal 1988; 8:5–6.

Moolgavkar SH, Dewanji A. Discussion of "From Mouse to Man: The Quantitative Assessment of Cancer Risks" by D.A. Freedman and H. Zeisel. Stat Sci 1988; 3:39–41.

Moolgavkar SH, Dewanji A, Venzon DJ. A stochastic two-stage model for cancer risk assessment. I: The hazard function and the probability of tumor. Risk Anal 1988; 8:383–392.

Moolgavkar SH. Some remarks on general relative risk regression models. Proc. Biopharmaceutical Section of ASA, 1988.

Moolgavkar SH. Biologically motivated two-stage model for cancer risk assessment. Toxicol Lett 1988; 43:139–150.

Moolgavkar SH, Venzon DJ. Confidence regions in curved exponential families: Application to matched case-control and survival studies with general relative risk function. Ann Stat 1987; 15:346–359.

Moolgavkar SH, Venzon DJ. Confidence regions for parameters of the proportional hazards model: A simulation study. Scand J Stat 1987; 14:43–56.

Lustbader ED, Moolgavkar SH. Some problems of inference in cohort studies. J Chron Dis 1987; 40(Suppl. 2):133–137.

Moolgavkar SH, Prentice RL. Discussion of the paper "Parameter Orthogonality and Approximate Conditional Inference," by D.R. Cox and N. Reid. JR Statist Soc 1987; B 49:34–35.

Moolgavkar SH, Venzon DJ. General relative risk models for epidemiologic studies. Am J Epidemiol 1987; 126:949–961.

Moolgavkar SH. Carcinogenesis modelling: From molecular biology to epidemiology. Ann Rev Pub Health 1986; 7:151–170.

Moolgavkar SH, Venzon DJ. Confidence regions for case-control and survival studies with general relative risk functions. In: Modern Statistical Methods in Chronic Disease Epidemiology. Proc. SIMS Conference. Moolgavkar SH, Prentice RL (eds), John Wiley, 1986.

Knudson AG, Moolgavkar SH. Inherited influences on susceptibility to radiation carcinogenesis. In: Radiation Carcinogenesis. Upton AC (ed), Elsevier/North Holland, 1986.

Prentice RL, Moolgavkar SH, Farewell VT. Biostatistical issues and concepts in epidemiologic research. J Chron Dis 1986; 38:1169–1183.

Moolgavkar SH. Hormones and multistage carcinogenesis. Cancer Surv 1986; 5:635–648.

Moolgavkar SH. Antioncogenes and cancer. pp. 19–30. In: Pathophysiological Aspects of Cancer Epidemiology. Mathe' G, Reizenstein P (eds), Pergamon Press, 1985.

Moolgavkar SH. Mutation and human cancer. pp. 31–38. In: Pathophysiological Aspects of Cancer Epidemiology. Mathe' G, Reizenstein P (eds), Pergamon Press, 1985.

Venzon DJ, Moolgavkar SH. Cohort analysis of malignant melanoma in five countries. Am J Epidemiol 1984; 119:1, 62–70.

Stevens RG, Moolgavkar SH. A cohort analysis of lung cancer and smoking in British males. Am J Epidemiol 1984; 119:624–641.

Stevens RG, Moolgavkar SH. Malignant melanoma: Dependence of site-specific risk on age. Am J Epidemiol 1984; 119:890–895.

Moolgavkar SH, Lustbader ED, Venzon DJ. A geometric approach to non-linear regression diagnostics with application to matched case-control studies. Ann Stat 1984; 12:816–826.

Stevens RG, Moolgavkar SH. Smoking and cancer in Britain. Proc. 5th World Conference on Smoking and Health, 1984.

Moolgavkar SH. Some comments on the resources at RERF. pp. 274–279. In: Utilization and Analysis of Radiation Effects Research Foundation Data. Proc. SIMS Conference. Prentice RL, Thompson DJ (eds), SIAM, 1984.

Lustbader ED, Moolgavkar SH, Venzon DJ. Tests of the null hypothesis in case-control studies. Biometrics 1984; 1017–1024.

Moolgavkar SH. Model for human carcinogenesis: Action of environmental agents. Environ Health Perspect 1983; 50:285–291.

Moolgavkar SH. A model for human carcinogenesis: Hereditary cancers and premalignant lesions. Proc. 7th Chicago Cancer Symposium, Cancer: Etiology and Prevention. Crispen RG (ed), Elsevier Science Publishing Co., Inc., 1983.

Stevens RG, Moolgavkar SH, Lee JAH. Temporal trends in breast cancer. Am J Epidemiol 1982; 115:759–777.

Moolgavkar SH. Risk assessment using vital data. pp. 175–192. In: Environmental Epidemiology: Risk Assessment. Proc. SIMS Conference. Prentice RL, Whittemore AS (eds), SIAM, 1982.

Moolgavkar SH, Knudson AG. Mutation and cancer: A model for human carcinogenesis. JNCI 1981; 66:1037–1052.

Moolgavkar SH, Stevens RG. Smoking and cancers of bladder and pancreas: Risks and temporal trends. JNCI 1981; 67:15–23.

Stevens RG, Lee JAH, Moolgavkar SH. No association between oral contraceptives and malignant melanoma. N Engl J Med 1980; 302:966.

Moolgavkar SH. The Neyman-Scott carcinogenesis model for low-dosage extrapolation. Math Biosci 1980; 50:155–156.

Moolgavkar SH, Day NE, Stevens RG. Two-stage model for carcinogenesis: Epidemiology of breast cancer in females. JNCI 1980; 65:550–569.

Moolgavkar SH. Multistage models for carcinogenesis. JNCI 1980; 65:25.

Moolgavkar S, Stevens RG, Lee JAH: The effect of age on the incidence of breast cancer in females. JNCI 1979; 62:493–501.

Moolgavkar SH, Venzon DJ. Two-event model for carcinogenesis: Incidence curves for childhood and adult tumors. Math Biosci 1979; 47:55–77.

Stevens RG, Moolgavkar SH. Estimation of relative risk from vital data: Smoking and cancers of the lung and bladder. JNCI 1979; 63:1351–1357.

Moolgavkar S, Lee JAH, Hade RD. Comparison of age-specific mortality from breast cancer in males in the U.S. and Japan. JNCI 1978; 60:1223–1225.

Moolgavkar S. The multistage theory of carcinogenesis and the age distribution of cancer in man. JNCI 1978; 61:49–52.

Moolgavkar S. The multistage theory of carcinogenesis. Int J Cancer 1977; 19:730.

Jarabak R, Colvin M, Moolgavkar S, Talalay P. Δ5-3-ketosteroid isomerase of Pseudomonas Testosteroni. pp. 642–651. In: Methods in Enzymology, Vol. XV. Clayton RB (ed), Academic Press, NY, 1970.

Books

Moolgavkar SH, Krewski D, Zeise L, Cardis E, Moller H (eds). Quantitative estimation and prediction of human cancer risk. IARC Scientific Publications, Volume 131, 1999.

Moolgavkar SH (ed). Scientific issues in quantitative cancer risk assessment. Birkhauser Boston, 1990.

Moolgavkar SH, Prentice RL (eds). Modern statistical methods in chronic disease epidemiology. John Wiley, 1986.

Tobacco Smoking. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. IARC, Volume 38, Lyon, 1986 (member of the working group).

Selected Invited Presentations

Moolgavkar SH. Clonal expansion and carcinogenesis. International Conference on Systems Biology in Radiation Carcinogenesis, Munich, Germany, February 2007.

Moolgavkar SH. Epidemiology of colon cancer. AEK Cancer Congress, Frankfurt, Germany, February 2007.

Moolgavkar SH. Multistage carcinogenesis and epidemiologic studies of cancer. Distinguished Seminar Series, Fox Chase Cancer Center, PA, October 2005.

Moolgavkar SH. Multistage carcinogenesis and lung cancer prevention. IARC Seminar Series, Lyon, France, July 2004.

Moolgavkar SH. Radiation-induced gestational mutations and cancer. COSPAR meeting, Paris, France, July 2004.

Moolgavkar SH. Multistage carcinogenesis and radiation risk assessment. International Congress of Radiation Research, Brisbane, Australia, August 2003.

Moolgavkar SH. Cancer models and risk assessment. Environmental Mutagen Society, Annual Meeting, Miami, May 2003.

Moolgavkar SH. Methodological issues in time-series analyses of air pollution data. Meeting the Environmental Challenge of the 21st Century, Monterey, CA, March 2003.

Moolgavkar SH. Multistage carcinogenesis and risk assessment. International Biometrics Conference, Homburg, Germany, March 2001.

Moolgavkar SH. Multistage models of carcinogenesis: historical perspective, overview, implications for radiation carcinogenesis. International Workshop on Mathematical Models in Radiation Carcinogenesis, Kyoto, March 2001.

Moolgavkar SH. Modeling altered hepatic foci: issues and outstanding problems. 6th European Meeting on Hepatocarcinogenesis, Vienna, September 1999.

Moolgavkar SH. Intermediate lesions in carcinogenesis. Netherlands Institute for Health and the Environment Seminar Series, 1997.

Moolgavkar SH. Multistage model for lung cancer. International meeting of the Bernoulli Society, Calcutta, India, 1997.

Moolgavkar SH. Stochastic cancer models: Application to analyses of solid cancer incidence in the cohort of A-bomb survivors. Keynote Speaker, International symposium on low-dose and low-dose-rate radiation, Stratford-on-Avon, UK, 1997.

Moolgavkar SH. Stochastic models for estimation and prediction of cancer risk. International Symposium on Statistics in the Environment, Enschede, The Netherlands, 1997.

Moolgavkar SH. Time-series analyses of air pollution data. International Symposium on Health Effects of Particulate Air Pollution, Prague, 1997.

Moolgavkar SH. Multistage carcinogenesis, benzene exposure and leukemia risk. Berkeley Symposium on Benzene and Leukemia, Napa Valley, 1996.

Moolgavkar SH. Mutations and cell proliferation in cancer risk assessment. AACR International Workshop on Risk Assessment, Whistler BC, 1994.

Moolgavkar SH. Analysis of altered foci in rodent hepatocarcinogenesis experiments. European Toxicology Meeting, Mainz, Germany, 1993.

Moolgavkar SH. Biologically-based cancer risk assessment. International Symposium on Quantitative Risk Assessment, Research Triangle Park, NC, 1993.

Moolgavkar SH. Analysis of altered foci in rodent hepatocarcinogenesis experiments. International Workshop on Mouse Liver Tumors, Washington DC, 1992.

Moolgavkar SH. Cancer models and low-dose extrapolation of risk. Workshop on Risk Assessment and Low Dose Extrapolation, Zurich, Switzerland, 1992.

Moolgavkar SH. Cell proliferation and carcinogenesis. International Conference on Cell Proliferation in Carcinogenesis, NIEHS, North Carolina, 1992.

Moolgavkar SH. Multistage carcinogenesis and risk assessment. International Toxicology Conference, Rome, Italy, 1992.

Moolgavkar SH. A population perspective on multistage carcinogenesis. Princess Takamatsu Cancer Congress, Tokyo, Japan, 1991.

Moolgavkar SH: Cancer models. International Workshop on Biophysical Modelling of Radiation Carcinogenesis, Padua, Italy, 1991.

Moolgavkar SH. Carcinogenesis models: An overview. Hanford Symposium on Health and the Environment, Battelle PNL, Richland, WA, October 1990.

Moolgavkar SH. Analyses of altered foci in rat hepatocarcinogenesis experiments. University of Vienna Cancer Center, Vienna, Austria, July 1990.

Moolgavkar SH. Multistage models of carcinogenesis. University of Tübingen Seminar Series, Tübingen, July 1990.

Moolgavkar SH. Analyses of intermediate lesions in experimental carcinogenesis. German Cancer Research Center, Heidelberg, Germany, June 1990.

Moolgavkar SH. Analyses of altered foci in rat hepatocarcinogenesis experiments. BASF, Toxicology Group, Mannheim, 1990.

Moolgavkar SH. Cell proliferation and carcinogenesis. International Cancer Congress, Hamburg, 1990.

Moolgavkar SH. Multistage carcinogenesis. University of Pittsburgh, Department of Biostatistics Seminar Series, 1990.

Moolgavkar SH. Analysis of altered foci in hepatocarcinogenesis experiments. McArdle Laboratory, University of Wisconsin, Madison, WI, 1989.

Moolgavkar SH. Biologically-based cancer risk assessment. Society for Risk Analysis, Annual Meeting, San Francisco, CA, 1989.

Moolgavkar SH. Multistage carcinogenesis and radiation risk assessment. Radiation Research Society, Annual Meeting, Seattle, WA, 1989.

Moolgavkar SH. The role of somatic mutations and cell replication kinetics in quantitative cancer risk assessment. International Conference on Chemically Induced Cell Proliferation: Implications for Risk Assessment, Austin, TX, 1989.

Moolgavkar SH. Two mutation model for carcinogenesis: Relative roles of somatic mutations and cell proliferation in determining risk. SIMS Conference on Scientific Issues in Quantitative Cancer Risk Assessment, Alta, Utah, 1989.

Moolgavkar SH. Cancer models and risk assessment. NATO Workshop on Biologically-based Methods for Cancer Risk Assessment, Corfu, Greece, June 1988.

Moolgavkar SH. A two-stage model for carcinogenesis and its implications for risk assessment. University of Nebraska Medical Center, May 1988.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Risk Assessment Workshop, Washington, DC, March 1988.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Health and Welfare, Ottawa, Canada, March 1988.

Moolgavkar SH. Curvature and inference in exponential families: Application to Relative Risk Regression Models. Carleton University, Ottawa, Canada, March 1988.

Moolgavkar SH. Cox regression for the innocent bystander. Fox Chase Cancer Center Seminar, Philadelphia, PA, December 1987.

Moolgavkar SH, Prentice R. Modern statistical methods in chronic disease epidemiology. Biopharmaceutical Section of ASA (tutorial and short course), Newark, NJ, December 1987.

Moolgavkar SH. Biologically motivated two-stage model for carcinogenesis. 17th Conference on Toxicology, Wright-Patterson Air Force Base, Dayton, OH, November 1987.

Moolgavkar SH. Two-stage model for carcinogenesis. University of Wisconsin Seminars, "Curvature and Inference in Exponential Families: Application to Relative Risk Regression Models," Department of Human Oncology, Madison, OH, November 1987.

Moolgavkar SH. Two mutation model for cancer risk assessment. EPA Toxicology and Microbiology Seminar Series, Cincinnati, OH, October 1987.

Moolgavkar SH. Origin invariant relative risk functions: multi-stage models for cancer risk assessment. American Statistical Association Annual Meeting, San Francisco, CA, August 1987.

Moolgavkar SH. Biologically-based carcinogenesis models for risk assessment. Risk Assessment Workshop, Washington, DC, March 1987.

Moolgavkar SH. Two-stage model for carcinogenesis: implications for risk assessment. Symposium on Quantitative Assessment of Cancer Risk, Washington, DC, February 1987.

Moolgavkar SH. A cohort analysis of smoking and cancers of the lung, bladder and pancreas. School of Public Health grand rounds, Department of Biostatistics Seminar on General Relative Risk Regression Models for Epidemiologic Studies, University of Pittsburgh, Pittsburgh, PA, January 1987.

Moolgavkar SH. Two-stage model for carcinogenesis and the IPI protocol. Battelle PNL, Richland, WA, 1986.

Moolgavkar SH. Modern statistical methods in chronic disease epidemiology. SIMS conference, Alta, UT, June 1985.

Moolgavkar SH. Time related factors in cancer epidemiology. NIH International Symposium, April 1985.

Moolgavkar SH. General relative risk models for case-control studies. Johns Hopkins University, School of Public Health, Baltimore, MD, 1985.

Moolgavkar SH. Stochastic models for carcinogenesis and risk assessment. EPA, Washington, DC, 1985.

Moolgavkar SH. Risk assessment using vital data. SIMS Conference on Environmental Epidemiology and Risk Assessment, Alta, UT, June 1982.

Selected Professional Activities

- Consultant, Fox Chase Cancer Center
- Consultant, Health and Welfare, Canada
- Consultant, University of Nebraska Medical Center
- Member, IARC (International Agency for Research on Cancer) working group on Tobacco Smoking
- Member, NIH Special Study Section for Biometry
- Member, NSF panel to review scientific bases of risk assessment methodologies
- Member, External Science Advisory Board, RISC-RAD project of the European Union, ongoing

- Member, External Science Advisory Board, California Air Resources Board, ongoing
- Invited Expert, Workshop on Mechanisms of Fiber Carcinogenesis, IARC, Lyon, France, November, 2005
- Area Editor for Health and Environment, *Risk Analysis—An International Journal*, January 2000–present
- Senior Editor of monograph *Quantitative Estimation and Prediction of Cancer Risk* IARC Scientific Publications, No. 131, 1999
- Co-chairman, International Conference on Mathematical Models in Cancer, Park City, Utah, 1998
- Member, Health Effects Institute Expert Panel for re-analyses of critical air pollution studies, 1997–2000
- Member, Working Group on quantitative estimation and prediction of cancer risk, IARC, Lyon, 1993
- Member, Scientific Advisory Panel to the CIIT Centers for Health Research, 1992–2005
- Member, Scientific Advisory Panel to review the EPA Dioxin Health Assessment document, 1992
- Member, Scientific Advisory Panel to review Risk Assessment program of the National Center for Toxicologic Research, 1992
- Organizer and Chair, SIMS conference "Scientific Issues in Quantitative Cancer Risk Assessment", held in Snowbird, Utah, June 1989
- Member, Advisory Committee to review risk assessment program of Armstrong Laboratories, Wright-Patterson Air Force Base, 1987
- Member, External Scientific Committee to review the program of the Radiation Epidemiology Branch, NCI, 1987
- Co-chairman of SIMS conference "Modern Statistical Methods in Chronic Disease Epidemiology" held in Alta, Utah, in June 1985
- Session Chairman at International Symposium: "Time Related Factors in Cancer Epidemiology," held at NIH in April 1985

Testifying History 2002-2007

I have offered expert testimony in the following matters

2007 – Depositions

Larry Vucelich v. Ford Motor Company, General Motors Corp., Daimler-Chrysler Corp., et al. Circuit Court, Kanawha County, West Virginia.

Kenneth Hickman v. Union Carbide Corp., et al., Circuit Court, Kanawha County, West Virginia.

2006 – Trial Testimony

Carl & Joyce Holbrook v. Bondex International et al., King County, Washington

Rebekah Price v. Borg Warner et al. Superior Court of the State of California, Alameda County.

2006 – Depositions

United Sates et al. v. American Electric Power Service Corp. et al., Seattle, WA

Rebeckah Price v. Borg Warner et al. Superior Court of the State of California, Alameda County.

Carl & Joyce Holbrook v. Bondex International et al., King County, WA.

Joseph & Yolanda Tizcareno v. Burns Intnl. Services Corp Borg Warner et al., Los Angeles Superior Court, CA

Caroline Hicks v. American Asbestos Co. et al. San Francisco Superior Court, CA

Thomas & Elizabeth Halsema v. Allied Packing et al. Alameda County Superior Court, CA

2005 – Depositions

Samuel Gates v. A-1 Clultch Co. et al. San Francisco Superior Court, CA

Lewis Zavacky v. Dana Corporation et al. Cuyahoga County, Ohio.

2004 – Depositions

United States et al. v. Ohio Edison et al., Washington D.C.

2002 – Depositions

United States et al. v. W.R. Grace (cost recovery action), Denver, CO.